VOL. XXIV. September, 1897. No 9.

THE
Journal
OF
Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

Twenty-third Annual Meeting, held at St. John's Parish Hall, Washington, D.C., May 4th, 5th, and 6th, 1897.

The President, Dr. M. A. Starr, in the chair.

AUDITORY APHASIA.

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The term auditory aphasia, used to designate the sum total of defects in the use of language caused by a lesion of the auditory centre for words, or of the fibres immediately conected with it, is to be preferred to the other names used for the same group of sypmtoms. It is more definite than sensory aphasia, which includes the symptoms caused by injury of either the auditory or visual centre, or of both; and on the other hand, it is far more comprehensive than the term word-deafness, which applies to only one, although the most important one, of a number of related symptoms.

The following case is typical of the clinical aspect of auditory aphasia:

J. W. Long, aged 70, was very healthy during middle life, but after a severe attack of typhoid fever, in 1882, he suffered a great deal from chronic bronchitis and asthma. During the last week of March, 1896, on account of dyspnea, he passed most of the time at night in a chair, and his feet and ankles were somewhat swollen. On the morning of March 31st he had risen from his chair and was putting on his shoes, when he suddenly fell, unconscious. The physician who saw him an hour later found him still unconscious, with the right face, arm and leg paralyzed. The pulse was rapid and weak, but responded well to alcohol and digitalis, under the influence of which consciousness rapidly returned and the swelling of the feet disappeared, but then it was found that the patient could neither speak intelligibly nor understand what was said to him.

When I examined him, six days later, the hemiplegia, although still evident, had to a great extent disappeared, and in a few weeks no trace of it could be detected. From that time to the present (April, 1897) his general health has been good, but the speech defect to be described has shown no marked change.

Word-deafness is almost total. The patient cannot be made to put out the tongue or perform the simplest movement without being shown what is expected of him, nor does he give the slightest sign of understanding simple questions concerning his name, age and residence. Salutations are the only expressions that seem to be understood; thus when I approach him and say, "How are you today?" he replies quite naturally, "Very well, I thank you." But on being asked, "Do you understand what I am saying?" he replies, "How?" and on the question being repeated as distinctly as possible, he says vaguely, "No, can't say that I do." Hearing for the watch and for ordinary noises is quite good.

Spontaneous speech is copious and fluent, and many words are correctly articulated, but many others are mutilated and many sounds intended for words are utterly in-

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talking quite well, his intonations are natural and the expressions of his face are intelligent. He cannot be made to repeat words said to him, but when he understands what is wanted he can count up to about eight. He cannot be made to say the days of the week or the months of the year.

Acuity of vision is good. The patient easily finds his way about the city, recognizes persons at a distance and comprehends any visible change in his surroundings. Examination of the fields is difficult, but there seems to be no considerable defect; hemianopsia can certainly be excluded. On being shown a number of familiar objects, he very rarely ventures to name one, and if he does, the name is wrong. The aid of other senses makes no difference. When shown three familiar objects he cannot select the one named. His ability to use tools and other objects is not impaired, and he seems to have an intelligent understanding of pictures.

Comprehension of print and writing is lost, excepting his own name. Formerly he read a great deal, but now, on being urged to read, he replies with one of his few intelligible sentences, "I've lost all that." On being shown his name incorrectly written, "G. W. Long," he instantly recognizes it and points out the error, slowly writing a J in place of the G. On attempting to read it aloud, he reads, "J. W. Cullerdly;" the name "Grover Cleveland," he reads "Cupboard Crawford," and all his reading aloud is markedly paralexic. Nevertheless, he can correctly name the letters in many words, and after spelling a word he comes much nearer to pronouncing it, although he does not appear to understand it any better.

Attempts to read the time from a watch never succeed completely, although the minutes are sometimes read correctly. For example, the hands indicating 3.20, he says, "Twenty minutes after—" and then follows a jargon. Trying again, he says, "Fifteen minutes after—, fifteen minutes after three—. Don't know when in my life—," followed again by jargon.

He can write his own name correctly, but in carving it on a stick, he made it "J. W. Loog." Spontaneous and dictational writing are totally lost. Copying script is fairly well done, with the occasional doubling or substitution of a letter. Words in Roman capitals are copied into script nearly as well. On being given a very simple problem in addition, he sets down some figures for the sum, but they are absurdly incorrect.

As there are many unsettled questions concerning aphasia in general, and this form in particular, and as the final appeal must always be to a comparison of clinical records and autopsy findings, I have studied and tabulated all the reports accessible to me of well-observed cases in which an autopsy showed the auditory centre for words, or its immediate neighborhood, to be damaged, without involvement of the motor-speech centre or of the visual centre for words.

It is now generally agreed that the auditory centre for words is situated in the posterior two-thirds of the first temporal convolution, on the left side in normal individuals, but on the right in left-handed ones. A more precise location is highly desirable, and this seems to have been furnished by Flechsig, who, at a period when the auditory fibres are conspicuous from having just received their medullary sheaths, has traced most of them to two transverse gyri on the inner or Sylvian surface of the posterior part of the first temporal convolution. In these two gyri, especially the anterior one, and in the sulcus between them, he, accordingly, locates the auditory centre. This is quite consistent with the autopsy records as far as they go, but most observers do not seem to have paid any particular attention to the inner surface of the convolution.

Altogether seventeen cases have been found which are regarded as fulfilling the necessary conditions. Five are included in Starr's list (The Pathology of Sensory Aphasia, Brain, July, 1889); the others, with one exception, have been published since. Cases in which the lesion extends backward no further than the supramarginal gyrus are included, because such an extension does not seem to alter the clinical picture of auditory aphasia; but cases in which the lesion extended further backward have been rejected, on account of the uncertainty as to whether some of the symptoms have not been due to involvement of the visual centre, or of the fibres in immediate relation with it. In Case 9 the motor speech centre was found to be somewhat atrophied, but the case is included because Mills regarded this atrophy as a result of the long-continued disuse of the motor-speech apparatus which was secondary to the auditory lesions.

It must be confessed that some of these cases leave much to be desired as to fullness and accuracy in both the clinical and post-mortem records, and also that, owing to my distance from a large medical library, I have not had access to the original report in some cases, but have been compelled to rely on the best abstract I could find. Still, taking the cases together, with every due allowance for possible omissions or inaccurate statements, they furnish material for some valuable conclusions.

The cases are so tabulated that each one may draw his own conclusions from them, but a brief summary of the symptoms and of the writer's inferences from them may not be out of place.

Word-deafness existed to a marked degree in all these cases, although it should be noted that in Case 5 it was part of complete deafness due to the bilateral auditory lesion. There can be no doubt that word-deafness is the most characteristic symptom of a lesion of the auditory centre for words, and that it is a constant one.

Verbal amnesia, or impairment of the power to recall words to mind, is noted in eleven cases; in six nothing definite is said on this point, but in three of them (5, 8 and 15) it is safe to infer a degree of verbal amnesia corresponding to the degree of paraphasia, and in another (Case 3) the patient spoke so little that we would naturally infer considerable inability to recall words. In Case 10 spontaneous speech was said to be good, and if this was correct the recollection of words must also have been good, but the

CASES OF AUDITORY APHASIA WITH AUTOPSY.

REPORTER AND REFERENCES.	LESION.	COMPREHENSION OF SPEECH.	POWER TO RECALL WORDS.	SPONTANEOUS SPEECH.	IMITATIVE SPEECH.	READING ALOUD.	COMPREHENSION OF WRITING AND PRINT.	SPONTANEOUS WRITING.	COPYING.
1. Wernicke, Der aphasische Symptomencomplex, Breslau, 1874, Case 10.	Abscess of greater part of left temporal lobe. White matter of T_1 softened; cortex normal.	Impaired.	Much impaired.	Paraphasia.		Paralexia.			
2. Kussmaul, Ziemssen's Cyclop., Vol. XIV., p. 764.	Softening of T_1 and T_2, right, mainly cortical. Patient left-handed.	Impaired.	Impaired.	Paraphasia.		Good.	Lost.	Lost.	
3. Giraudeau, Rev. de Médecine, 1882, quoted in detail by Bernard, cited by Starr.	Sarcoma destroying posterior part of T_1 and T_2, left, mainly cortical.	Lost.		Good, but spoke very little.	Lost.	Good (tests rather meagre.)	Good (after reflection.)	Good.	
4. Claus, Irrenfreund, 1883, p 82, cited by Starr.	Softening of T_1 and T_2, left; cavity occupying all of left temp. lobe except anterior two thirds.	Lost.		Fluent; no motor loss.					
5. Wernicke and Friedländer, Fortsch. d. Medicin, I., 1883, No. 6.	Gumma and softening of posterior two-thirds of T_1 and T_2 and all projection fibres of temp. lobe, left. Also of A. Sm and posterior part of T_1, right.	Deafness (owing to bilateral auditory lesion.)		Paraphasia.	Lost.				
6. Sepilli, Rivista Sperimental di Freu. e di Med. Leg. 1884, quoted in detail by Amidon, N. Y. Med. Journal, Feb. 14, 1885.	Softening of T_1, T_2 and Sm, left; cortical.	Lost.	Much impaired.	Jargon paraphasia.	Fair.				
7. Hitzig, Cong. f. Inner Med., 1887, p. 166.	Softening of posterior two-thirds of T_1 and T_2, left.	Lost.	Lost.	Paraphasia.					
8. Heubner, Neurologisches Centralblatt, 1889, p. 697.	Softening 27mm in diameter at junction of Sm and T_1, prolonged forwards 65mm between T_1 and T_2, left; mainly cortical.	Lost.	Much impaired.	Lost.	Good.	Good.	Lost.	Paragraphia.	

9. Mills, Brain, Winter Part, 1891.	Softening T1 and T2, bilateral; left lesion older than right. First stroke 13 yrs., second 9 yrs. before death; symptoms noted in interval.	Lost.		Paraphasia.			Good.	Good.	
10. Pick, Archiv für Psychiatrie, XXIII., 3.	Softening T1 and Sm, left, mainly cortical; T1, T2 and insula, right.	Lost.		Good (slight paraphasia.)	Lost.	Good.	Good.	Good.	
11. Ascher, Allg. Zeitschr. f. Psych., XLIX., p. 256. Resumé in Neur. Centr., 1892, p. 688.	Focal degeneration T1, left. Diffuse degeneration of paretic dementia.	Much impaired.	Much impaired.	Paraphasia (used very few words.)	Good.	Good.	Lost.	Much impaired.	Fair.
12. Fraser, Glasgow Medical Jour., 1893. Resumé in Neur. Centr., 1893, p. 303	Atrophy T1 and gyrus opercularis, left.	Lost.	Much impaired.	Paraphasia.	Lost.	Good except for difficult words			
13. Leva, Virchow's Archiv, CXXXII., 2. Resumé in Neur. Centr., 1893, p. 786.	Cyst 3cm in diameter occupying T1 and T2, left. Slight focus of softening in foot of F2, left.	Impaired.	Impaired.	Impaired.	Good,	Impaired.	Lost.	Lost.	Impaired.
14. Mader, Wiener Med. Blätter, 1894, No. 8. Resumé in Neur. Centr., 1894, p. 700.	Softening T1, T2 and posterior part of insula, left.	Lost.	Much impaired.	Jargon paraphasia.	Lost.	Paralexia.	Lost.		
15. Worcester, Amer. Jour. Insanity, Oct., 1896, Case 3.	Disorganization of T1, T2 and T3, in posterior two-thirds, left, by bullet wound. Superficial track of ball over convolutions from temporal bone to apex of frontal lobe.	Marked word-deafness.	Much impaired.	Paraphasia.	Good.	Paralexia.		Lost except for names of self and wife.	Paragraphia.
16. Worcester, l. c., Case 4.	Brain atrophied. T1 left, very thin, yellowish and leathery. Convolutions elsewhere, normal in color, cortical layers distinct.	Lost.		Jargon paraphasia.					
17. Worcester, l. c., Case 5.	Softening of superior surface of posterior two thirds of T1 left, extending to the neighboring part of insula; also of foot of F1, right.	Marked word-deafness.	Much impaired.	Paraphasia.					

EXPLANATION: T1, T2 and T3—first, second and third temporal convolutions; F1, F2 and F3—frontal convolutions; Sm—supra-marginal gyrus; A—angular gyrus.

report indirectly indicates a slight degree of paraphasia, which implies a corresponding degree of verbal amnesia. It is safe to say that verbal amnesia exists in about five-sixths of all cases of auditory aphasia, and it is very probable that careful tests would show it to occur in all cases except those in which the lesion is purely subcortical.

Spontaneous speech was lost in one case (8) and impaired (paraphasic) in thirteen; in Case 3 it was said to be good, but the patient spoke very little; in Case 4 it is described as fluent with no motor loss. Of Case 10 it is said that only occasionally a substitution of words occurred, indicating a slight degree of paraphasia.

The last case, being one of almost pure word-deafness and the only one with autopsy that I have been able to find, has a most important bearing on the theory of subcortical auditory aphasia. As is well known to all students of aphasia, Wernicke, Lichtheim and others explain the verbal amnesia, paraphasia, loss of imitative speech, paralexia, loss of comprehension of print and paragraphia, which are commonly associated with word-deafness, to be due to a loss of the normal influence of the auditory centre upon the other centres concerned in speech, and such a combination of symptoms is regarded as proof that the lesion is cortical. On the other hand, word-deafness not accompanied by the other symptoms is explained on the hypothesis of a lesion of the auditory fibres on their way to the auditory centre, the centre itself, although deprived of impulses from the ears, retaining its memories of sound and thus being able to exert its normal control over the other centres. The symptoms of Case 10 accordingly led Pick to diagnosticate a subcortical auditory lesion, but at the autopsy a cortical lesion was found, only slightly invading the white matter. Possibly a close examination of the auditory centre, as defined by Flechsig, with the white matter immediately underneath would have removed this disappointing discrepancy, but, until other cases of the kind come to autopsy, we must accept this case as almost disproving the validity of the theoretical distinction between cortical and subcortical auditory aphasia.

The retention or loss of imitative speech is noted in ten cases. This function is worthy of careful attention, as its retention is the only thing that distinguishes transcortical from cortical auditory aphasia in Lichtheim's scheme. Case 8, if it stood alone, would be a brilliant illustration of the theory of transcortical auditory aphasia, for its symptoms were word-deafnes, verbal amnesia, loss of spontaneous speech, loss of comprehension of print and paragraphia, with retention of imitative speech, just as the theory requires; and the lesion was so situated in the supramarginal gyrus and in the sulcus between the first and second temporal convolutions as not to destroy the auditory centre, but to cut off its communications upward. backward and downward. Hence if we supose the association fibres from the auditory to the motor-speech centre to pass directly forward by way of the insula, they must have been spared, and everything about the case would be well explained. But in Cases 6, 11, 13 and 15, in which imitative speech is recorded as " fair " or " good," while the other symptoms were similar to those of Case 8, the lesion appears to have been cortical rather than transcortical. Moreover it is by no means certain that in Case 8 the path from the auditory to the motor-speech centre was intact, for it is only an assumption that this path passes directly forward—an assumption not justified by a study of the effects of lesions of the insula. On the contrary it seems to me more probable that this path takes a curved course, passing around the posterior extremity of the fissure of Sylvius, and that the " *Leitungsaphasie* " of Wernicke and Lichtheim is more likely to be found as a symptom of lesion of the supramarginal gyrus than of the insula. It must be remembered, too, that the retention of imitative speech accompanying word-deafness and paraphasia may be explained as due to a partial lesion of the auditory centre itself. A word actually spoken stimulates the auditory centre more powerfully than the corresponding idea, hence imitative speech may be good when spontaneous speech is paraphasic; and the auditory centre can arouse the motor-speech centre more easily than it can the centres for ideas (proved by the existence of echolalia without understanding in various stages of incomplete development and impairment of brain function), hence in case of partial damage to the auditory centre imitative speech may be good when the understanding of speech is impaired.

Reading aloud was tested in ten cases . In four of these there was decided paralexia corresponding to the paraphasia; in Case 12 there was a slight paralexia (for difficult words only) along with marked paraphasia; in Case 10 reading aloud was good and paraphasia scarcely appreciable; in Case 3 the patient read short sentences correctly, and there was no paraphasia; in Cases 2, 8 and 11 reading aloud was good, but the patients had no comprehension of what they read, and there was marked paraphasia. This ability to read aloud, without understanding and without the ability to speak correctly, is in itself a very remarkable phenomenon, and is in strong contrast to the paralexia of other cases. In the present state of knowledge no difference between the lesions of the two groups of cases can be assigned to explain why the patients of one group can read while the others can not, and the facts seem to laugh at every theory of the process of reading. Considering only the cases without paralexia, we would naturally infer that in reading aloud the association impulses must pass directly from the visual centre for words to the motor-speech centre by a path that is quite independent of the auditory centre. Considering only the cases of paralexia, we would infer, on the contrary, that in reading aloud the motor-speech centre is dependent upon guiding impulses from the auditory centre, just as it is in spontaneous speech.

I think the true inference from all these cases is that both in reading aloud and in naming a visible object two sets of association impulses pass forward from the visual centre, one set going directly to the motor-speech centre, exciting it to the utterance of words, but not always of the correct ones; another set going to the auditory centre, reviving the proper sound memories and exciting it to send a supplemental set of impulses to the motor-speech centre to insure the correct utterance. In some individuals the direct association between the visible words and the corresponding utterance has become so strong that it is of itself sufficient. In most individuals, however, the motor-speech centre will go astray, if not guided by the auditory centre, and in them an auditory lesion causes paralexia.

Possibly a similar explanation may apply to the occasional absence of paraphasia, and if so, it is evident, from the greater frequency of paraphasia, that the supplemental auditory impulses are more essential to spontaneous speech than to reading aloud. This is what we ought to expect. Letters being few and ideas many, each letter, by frequent repetition of the process, can become more strongly associated with its corresponding utterance than can each idea; and a centre, when excited by a sensation, sends out stronger association impulses than when excited by an idea.

The ability to understand print and writing was noted in eight cases. In five it was lost; in Case 3 it was said to be good after a little reflection, but does not appear to have been thoroughly tested; in Case 9, friends of the patient said it was good, but the physician did not have an opportunity to observe it for himself, and the testimony of an uncritical observer must be accepted with caution; in Case 10 it was undoubtedly good, corresponding to the ability to speak, read aloud and write correctly. Taken together these cases support the view of Wernicke and Lichtheim, that in the comprehension of written language the ideas are aroused by the auditory centre or, in other words, that the reader understands because he hears himself talk. I am convinced of the correctness of this view, but it must be admitted that there are anomalies which in the present state of knowledge do not admit of a satisfactory explanation.

Spontaneous writing was tested in eight cases. In five it was lost or impaired, the impairment being in the form of paragraphia. In Case 3, although the patient did not write much, spontaneous writing seems to have been better than spontaneous speech, quite contrary to the general rule in all forms of cortical aphasia. In Case 9 it was reported as good by the relatives, contrary to what we would expect, considering the existence of paraphasia and paralexia. In Case 10 it was good, in harmony with the correctness of spontaneous speech.

Copying was tested in only three cases. In Cases 13 and 15 it was impaired. Any decided impairment, not accounted for by the patient's general condition or by his distaste for copying what he does not understand, would justify the inference that there was more than an auditory lesion. In Case 13 a small lesion was found in the foot of the left second frontal convolution, and in Case 15 there was some involvement of the frontal lobe.

In summarizing the symptoms of auditory aphasia, we may say that word-deafness is constant; verbal amnesia and paraphasia are found in about five-sixths of the cases; probably a careful examination would show their presence in a larger proportion if not in all; imitative speech is sometimes retained, but more frequently lost or impaired, the difference probably depending upon the severity rather than upon the location of the lesion; reading aloud is often paralexic, but in many cases it is correct; comprehension of print was lost lost in the majority of cases tested, while in many of the severer cases no test seems to have been made; spontaneous writing was lost or paragraphic in most of the cases tested.

Does auditory aphasia constitute a fundamentally distinct type? Probably no one would for a moment question the fundamental difference between it and the ordinary type of motor aphasia, but there are eminent authorities who do not recognize any radical distinction between auditory and visual aphasia. Thus Mirallié (L'Aphasie Sensorielle, Paris, 1896, p. 53), speaking also for Dejerine, contends that there is only one variety of sensory aphasia and that its symptoms include both word-deafness and word-blindness. He admits that either of these symptoms may predominate over the other, but still holds that both can be detected in every case, and that apparent differences of type depend upon differences of degree and not of kind. It seems to me that the general adoption of such a doctrine would be an unnecessary step backward. It is, of course, understood that a lesion limited to the auditory sphere may at first cause visual symptoms and *vice versa*, but indirect and temporary consequences should not be confused with the direct and permanent effects of a lesion. And it is also understood that a large lesion in the Sylvian region usually affects both the visual and auditory spheres, but it is the effect of small and limited lesions that should be considered. If any one doubts that there are two perfectly distinct types of sensory aphasia, let him compare such a case as the one just reported, characterized by word-deafness, jargon paraphasia, paralexia, loss of comprehension of print, agraphia, ability to copy Latin letters into script and absence of any visual defect, with such cases of visual aphasia as those reported by Wilbrand,[2] Lissauer,[3] Dejerine,[4] Dejerine and Vialet,[5] Monakow[6] and Redlich.[7] In each of these cases in which the autopsy showed the visual sphere to be the only part of the language mechanism affected, comprehension of speech was good, spontaneous speech was good, or only slightly impaired, and there was right homonymous hemianopsia. In each of these cases there was naturally an inability to comprehend print, and as this inability is generally found in auditory aphasia, it may seem to be true that word-blindness is common to both forms of sensory aphasia. It must be remembered, however, that, reading being a complex process, alexia may be caused in three quite different ways, by motor-speech lesions, by auditory lesions and by visual lesions, and it is only to alexia caused by visual lesions that the term word-blindness should be applied. The patient whose case I have reported in this paper gave abundant evidence that his visual functions, including visual memory, were in a condition of normal activity. It is not necessary to assume any degree of word-blindness to account for his alexia; he could not read because he was word-deaf for internal as well as external speech.

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[2] Quoted by Freund, Arch. f. Psych. XX.

[3] Arch. f. Psych., XXI., 1.

[4] Mém. de la Soc. de Biol., 1892, *résumé* in Neur. Centr. 1892, p. 373.

[5] Bull. Méd., Aug., 1893, *résumé* in Neur. Centr. 1893, p. 787.

[6] Arch. f. Psych. XXIII. 3.

[7] Jahrb. f. Psych. u. Neur., XIII, 2. 3, *résumé* in Archives de Neurologie, Jan. 1896.

Disregarding minor arguments the reasons for insisting that there are two radically distinct types of sensory aphasia may be stated thus: Good observers have described cases of defect in the use of language due to a lesion of the angular gyrus or its immediate neighborhood, in which (after the temporary and indirect effects had passed away) no word-deafness could be detected, but permanent visual symptoms were clearly apparent; visual aphasia is the appropriate name for these defects. Contrasted with such cases are those forming the subject of this paper, whose lesion is in the posterior part of the first temporal convolution, and in which there is marked and persistent word-deafness without any persistent visual symptoms whatever; the speech defects of such cases are properly called auditory aphasia.

The impairment of all the ways of using articulate language, which is so evident in a typical case of auditory aphasia, is no doubt due to the fact that the auditory centre in the first language centre to be developed, and to its being essential to the normal development and cooperation of the other language centres. The understanding of spoken words is the first linguistic acquisition in childhood, and this depends upon the formation of association tracts between the auditory centre and the various centres concerned in ideation. Utterance soon follows as an imitative reflex, and throughout life it depends for accuracy upon association impulses from the auditory to the motor-speech centre. Writing is an arbitrary representation of spoken words by visual signs, and reading is a reconversion of these signs into words as uttered and heard, and, as destruction of the auditory centre interferes with both understanding and utterance, it must also interfere with reading and writing.

DISCUSSION.

Dr. Philip Zenner, of Cincinnati—I will merely mention a case which was interesting to me, the post-mortem of which I saw last Friday. The patient was one who had Jacksonian attacks, and these were the only symptoms, with the exception of double optic neuritis; the attacks being chiefly in the right side of the tongue and right side of the face, though generally attended or introduced by sensory symptoms in the hand or face. Several times he had heard noises during the attacks, and one attack, described to me by his mother and himself, perhaps a day or two after it occurred, was distinctly an attack of word-deafness, in conjunction with the other symptoms already spoken of. For instance: I think he wanted a handkerchief, but he said, "Give me that shirt;" and when his mother said to him, "Here is a handkerchief," he evidently did not understand what she said. He admitted the next day that he had heard what had been said, but had not understood the words. At the autopsy a tumor was found lying in the lower part of the fissure of Rolando, evidently resting upon the first temporal convolution. This is the only case of word-deafness occurring as a part of a Jacksonian attack that I have seen, and I cannot recall having read the report of a similar case.

Dr. Charles K. Mills, of Philadelphia—I would like to say with regard to my case, to which Dr. Pershing has referred, and which was presented before this Association some years ago, that I still have the specimen in a pretty good state of preservation, and a short time ago glanced at it again. The retroinsular convolutions, which I understand to be the transverse convolutions referred to by Dr. Pershing, in this specimen are very much attenuated, as is also the whole of the posterior third, or even more than a third, of the first temporal convolution.

The original lesion, the one which seemed to have caused the word-deafness, did not affect the retroinsular convolutions, but was situated in the posterior portion of the first temporal convolution and in the parallel fissure between it and the posterior portion of the second temporal.

In the general discussion of his subject I understand Dr. Pershing to take a position somewhat in opposition to that taken by Dejerine and others. With Dr. Pershing I believe that isolated lesions of the auditory centre, and similar isolated lesions of the visual centre, or a portion of them, will cause special and limited forms of aphasia, and will not result in an implication of all the forms of language.

Dr. Worcester, of Danvers, Mass.—I have had considerable experience with cases of this sort, and they are not infrequently admitted into hospitals for the insane, generally with an incorrect diagnosis, and I judge that not very infrequently the diagnosis is not corrected there. At last year's meeting of the American Psychological Society I read a paper embodying the results of my observations in full. I had at that time had four autopsies of cases of sensory aphasia. In two of them there was softening, and in one atrophy of the posterior part of the first left temporal convolution, and in the fourth there was a gunshot injury of the temporal lobe involving that portion of the convolution. In all these cases there was paraphasia, word-deafness, paralexia, and paragraphia—as far as there was ability to write at all; one patient had never learned to write. Two of them could sing tunes correctly, and the same was true of some other cases which have not yet come to autopsy. I have had altogether sixteen cases of this sort under my observation, and have had two autopsies since the date of the paper to which I referred, one of which was a case of softening.

In addition to this I had one quite interesting case of subcortical aphasia; it was the most complete case of aphasia that I ever saw. The man was a Swiss, and could not, so far as I could determine, speak a single word; the only articulate sound to which he gave utterance was something like "Sagasso," and he made use of this on all sorts of occasions. He had some comprehension of gesture, but none of speech. There was much dementia, and there was also complete uselessness of the right hand, but he was able to hobble about with the right leg. At the autopsy I found softening involving the whole of the external capsule, and the greater part of the nucleus lenticularis. The cortex seemed to be entirely unaffected.

Dr. Joseph Collins—Instead of the auditory speech centre being spread out over too large a territory by the modern localizationalist and the student of physiology, it seems to me that it is allocated to an area so small that it is entirely incompatible with the grand importance which this centre has in conditioning speech development and maintaining the integrity of this function when it is developed. We have centred our attention too much on the supertemporal convolution of the left side, and have gone about looking for the smallest lesion of this area that would produce word-deafness. It would be more in line with the most legitimate facts and speculations in brain physiology to study the relation of the superior temporal lobe of the right hemisphere to the genesis of speech, and its preservation when developed. There are many facts indicating that the superior temporal convolution of the right side plays a most important part in these two things, and that the so-called auditory centre in the middle of the left supertemporal convolution is merely an executive centre, and is controlled by the same forces that determine righthandedness, be they tribal, sociologic or cultivated.

Dr. Pershing—The view Dr. Collins takes is the very one that I wish to combat in my paper; but the evidence will not be appreciated unless these cases, in which the post-mortem showed that the disease was limited to the auditory centre, are compared with the cases of lesion in the visual sphere, which I cited, though not at length. I think that if these groups of cases are put side by side my position will be substantiated.

In discussing the relative effect of lesions of the auditory sphere and of the visual sphere, it must be remembered that the auditory centre is the fundamental language centre; the first to be developed. All the later acquisition of the language depends on the activity of the auditory centre. As we learn to speak, or to read, or to write, we constantly call the auditory memory into use; therefore, if the auditory memory is destroyed, all these ways of using language are affected. Not so with the visual centre. If we may trust the reports of well-observed cases of visual aphasia, a lesion limited to the visual sphere does not cause word-deafness. This fact, I think, entirely disproves the views of Dejerine and Mirallié.

Dr. J. J. Putnam, of Boston—I had the opportunity to observe with care, some years ago, a case in which the patient was a literary man, and was able to make careful observations of his own symptoms. He eventually became hemiplegic, but at this time he was seized suddenly with word-blindness; he took down his Shakespeare, and found that he could not understand a single line. This passed away, but afterwards recurred. It was not, however, associated with any word-deafness at that time, certainly not to any considerable degree.